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## Non Invasive Imaging

### ABNORMAL GLOBAL LONGITUDINAL STRAIN TO PREDICT OUTCOMES IN HYPERTROPHIC CARDIOMYOPATHY

Poster Contributions

Hall C

Sunday, March 30, 2014, 9:45 a.m.-10:30 a.m.

Session Title: Non Invasive Imaging: Left Ventricular Myocardial Strain Imaging-Clinical Applications

Abstract Category: 12. Heart Failure and Cardiomyopathies: Clinical

Presentation Number: 1174-38

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**Background:** Although global longitudinal strain (GLS) predicts cardiovascular morbidity and mortality in certain populations, data evaluating GLS and outcomes in hypertrophic cardiomyopathy (HCM) are limited. Currently, a combination of clinical and imaging factors are primarily used to risk stratify patients with HCM. As normal GLS values have recently been validated, we hypothesized that abnormal GLS provides prognostic value in patients with HCM.

**Methods:** A HCM database (N = 180) including clinical and phenotypic characteristics, routine standard 2-D echocardiography with Doppler imaging, and record of major adverse cardiac events (MACE) was retrospectively queried. MACE, defined as heart failure hospitalizations, sustained ventricular arrhythmias, or all cause death represented the primary outcome. Other relevant variables assessed in this analysis included history of atrial fibrillation, left ventricular wall thickness >30 mm, resting left ventricular outflow gradient >30 mm Hg, at least moderate mitral regurgitation, and systolic anterior motion of the mitral valve. GLS analysis was performed on-line on standard 2D apical 2-, 3-, and 4-chamber views with normal GLS defined as more negative than -15%. GLS as a predictor of MACE was assessed in a univariate and multivariate model with the variables listed above.

**Results:** 78 subjects with GLS and complete record of longitudinal follow-up were included in the final analysis. The cohort was 54% male and 44 ± 16 years old at the time of imaging. Mean ejection fraction was 61% ± 6.3 and 55% had abnormal GLS. At a mean follow-up of 22 ± 15 months, 19% (15/78) reached the primary endpoint. Of those with abnormal GLS, 30% (13/43) reached the primary endpoint compared to 6% (2/35) with normal GLS (p < 0.01). In univariate analyses, abnormal GLS was the only predictor of MACE (p = 0.017). In a backward stepwise multivariate logistic regression model, abnormal GLS remained the only independent predictor of MACE (OR 6.05; 95% CI 1.1 - 32.6, p = 0.038).

**Conclusions:** In this single center cohort of patients with HCM, abnormal GLS was an independent predictor of MACE. Further prospective and larger studies are warranted to validate these findings.